```
L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2007 ACS on STN
RN   59803-98-4  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   6-Quinoxalinamine, 5-bromo-N-(4,5-dihydro-1H-imidazol-2-yl)-  (CA INDEX
     NAME)
OTHER NAMES:
CN   5-Bromo-6-(2-imidazolin-2-ylamino)quinoxaline
CN   AGN 190342
CN   Brimonidine
CN   Bromoxidine
CN   UK 14304
CN   UK 14304-18
DR   109826-55-3
MF   C11 H10 Br N5
CI   COM
LC   STN Files:   ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS,
       BIOTECHNO, CA, CAPLUS, CASREACT, CBNB, CHEMCATS, CIN, CSCHEM, DDFU,
       DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IMSDRUGNEWS, IMSPATENTS,
       IMSRESEARCH, IPA, MEDLINE, MRCK*, PHAR, PROMT, PROUSDDR, PS, RTECS*,
       SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
         (*File contains numerically searchable property data)
     Other Sources:   WHO
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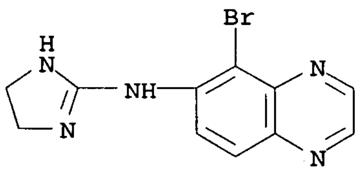

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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

             834 REFERENCES IN FILE CA (1907 TO DATE)
              12 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
             834 REFERENCES IN FILE CAPLUS (1907 TO DATE)
```

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FILE 'HOME' ENTERED AT 13:55:42 ON 06 JUL 2007

=> file reg
COST IN U.S. DOLLARS                                 SINCE FILE      TOTAL
                                                          ENTRY    SESSION
FULL ESTIMATED COST                                        0.21       0.21

FILE 'REGISTRY' ENTERED AT 13:55:50 ON 06 JUL 2007
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:    5 JUL 2007  HIGHEST RN 941372-96-9
DICTIONARY FILE UPDATES:   5 JUL 2007  HIGHEST RN 941372-96-9

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> E "BRIMONIDINE"/CN 25
E1             1     BRIMAC (CHARCOAL)/CN
E2             1     BRIMAC 216/CN
E3             1 --> BRIMONIDINE/CN
E4             1     BRIMONIDINE PURITE/CN
E5             1     BRIMONIDINE TARTRATE/CN
E6             1     BRIMSTONE/CN
E7             1     BRIMSTONE ACID/CN
E8             1     BRINALDIX/CN
E9             1     BRINASE/CN
E10            1     BRINASTRASE/CN
E11            1     BRINAZARONE/CN
E12            1     BRINDLEYITE/CN
E13            1     BRINDOXIME/CN
E14            1     BRINERDINE/CN
E15            1     BRINES, BITTERN/CN
E16            1     BRINEWATE SUPERFINE/CN
E17            1     BRINO/CN
E18            1     BRINOLASE/CN
E19            1     BRINOX/CN
E20            1     BRINROBERTSITE/CN
E21            1     BRINROBERTSITE
((AL3.83MG0.17)(SI7.83AL0.17)NA0.33(OH)4O20.7/2H2O)/CN
E22            1     BRINTOBAL/CN
E23            1     BRINZOLAMIDE/CN
E24            1     BRINZOLAMIDE HYDROCHLORIDE/CN
E25            1     BRIO/CN

=> S E3
L1             1 BRIMONIDINE/CN

=> DIS L1 1 IDE
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L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2007 ACS on STN
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       IMSRESEARCH, IPA, MEDLINE, MRCK*, PHAR, PROMT, PROUSDDR, PS, RTECS*,
       SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
         (*File contains numerically searchable property data)
     Other Sources:   WHO
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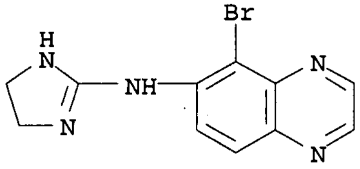

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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

             834 REFERENCES IN FILE CA (1907 TO DATE)
              12 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
             834 REFERENCES IN FILE CAPLUS (1907 TO DATE)


=> file caplus embase biosis medline
COST IN U.S. DOLLARS                              SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     7.80       8.01

FILE 'CAPLUS' ENTERED AT 13:56:51 ON 06 JUL 2007
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FILE 'BIOSIS' ENTERED AT 13:56:51 ON 06 JUL 2007
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FILE 'MEDLINE' ENTERED AT 13:56:51 ON 06 JUL 2007

=> s brimonidine or 59803-98-4
L2         5471 BRIMONIDINE OR 59803-98-4

=> s parkinsons(a)disease or parkinson's(a)disease
MISMATCHED QUOTE 'PARKINSON'S'
```

```
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s parkinsons(a)disease
L3          2860 PARKINSONS(A) DISEASE

=> s l2 and l3
L4             0 L2 AND L3

=> s parkinson's(a)disease
MISMATCHED QUOTE 'PARKINSON'S'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s alzheimers(a)disease
L5          2821 ALZHEIMERS(A) DISEASE

=> s l2 and l5
L6             0 L2 AND L5

=> s alpha(a)2(a)agonist
L7          6425 ALPHA(A) 2(A) AGONIST

=> s l3 and l7
L8             1 L3 AND L7

=> d ti au ab so py

L8    ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI    CNS targets for multi-functional drugs in the treatment of Alzheimers and
      Parkinsons diseases.
AU    Youdim, M. B. H. [Reprint Author]; Buccafusco, J. J.
AB    Patients with mild forms of dementia and age-related memory impairment
      have just begun to benefit from pharmacotherapy developed over the last
      several years.  However, current approaches do not significantly modify
      the course of neurodegeneration or of the aging process, and they offer
      limited and transient benefit to many patients.  The goal of this review
      is to summarize new potential approaches in which molecules have been
      developed expressly to target multiple brain systems for the treatment of
      memory and cognition impairment.  Some of these approaches include the
      development of single molecular entities that combine activity as
      cholinesterase inhibitors, muscarinic cholinergic M2 receptor antagonists,
      nicotinic acetylcholine receptor agonists, alpha(
      2)-adrenergic agonists, or monoamine oxidase inhibitors.  Many of
      the bi-functional compounds discussed have improved efficacy as cognitive
      enhancing agents and/or they offer potential for neuroprotection and
      disease modification.  It is likely that syndromes such as Alzheimer's
      disease will require multiple drug therapy to address the varied
      pathological aspects of the disease.  Even if the strategy of combining
      drugs with different therapeutic targets is workable, the development of
      multi-functional compounds will obviate the challenge of administering
      multiple single drug entities with potentially different degrees of
      bioavailability, pharmacokinetics, and metabolism.  Also, the
      simplification of the therapeutic regimen for individuals with AD who have
      difficulty with compliance is important.
SO    Journal of Neural Transmission, (APR 2005) Vol. 112, No. 4, pp. 519-537.
      CODEN: JNGSE8. ISSN: 0300-9564.
PY    2005


=> s neurodegeneration
L9         35579 NEURODEGENERATION
```

```
=> s l2 and l9
L10          4 L2 AND L9

=> d ti au ab so py 1-4

L10  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Methods for the treatment of ocular and neurodegenerative conditions in a
     mammal
IN   Ruiz, Guadalupe; Padillo, Edwin U.; Woldemussie, Elizabeth
AB   Method for the treatment of conditions including ocular and
     neurodegenerative conditions in a mammal using a composition which comprises an
     effective amount of an alpha (1) adrenoreceptor antagonist.
SO   PCT Int. Appl., 46pp.
     CODEN: PIXXD2
PY   2006
     2006

L10  ANSWER 2 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Method of using (2-imidazolin-2-ylamino)quinoxalines, especially
     brimonidine, in the treatment of dementia and Parkinson's disease
IN   Tatton, William G.; Wheeler, Larry A.; Gil, Daniel W.; Donello, John E.
AB   Methods of preventing or retarding the degeneration of neurons and/or
     methods for treating Alzheimer's disease or Parkinson's disease,
     comprising administering a therapeutically effective amount of
     brimonidine.
SO   PCT Int. Appl., 48 pp.
     CODEN: PIXXD2
PY   2004
     2004
     2004
     2004
     2005
     2005
     2005
     2006
     2005
     2006

L10  ANSWER 3 OF 4  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   α2B or α2B/2C Adrenoceptor agonists for the treatment of
     neurodegeneration
IN   Wheeler, Larry A.; Gil, Daniel W.; Donello, John E.
AB   The invention discloses methods using α2B or α2B/2C
     adrenoceptor agonists for preventing or retarding the degeneration of
     neurons.  Also disclosed are methods for treating Alzheimer's disease or
     Parkinson's disease through the administration of selective α2B or
     α2B/2C adrenoceptor agonists.
SO   PCT Int. Appl., 36 pp.
     CODEN: PIXXD2
PY   2004
     2004
     2004
     2004
     2005
     2005
     2005
     2006
     2007
     2005
     2006
     2006

L10  ANSWER 4 OF 4  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
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     reserved on STN
TI   Neuroprotection in glaucoma.
AU   Kaushik S.; Pandav S.S.; Ram J.
AB   Currently, glaucoma is recognised as an optic neuropathy.  Selective death
     of retinal ganglion cells (RGC) is the hallmark of glaucoma, which is also
     associated with structural changes in the optic nerve head.  The process
     of RGC death is thought to be biphasic: a primary injury responsible for
     initiation of damage that is followed by a slower secondary degeneration
     related to noxious environment surrounding the degenerating cells.  For
     example, retinal ishaemia may establish a cascade of changes that
     ultimately result in cell death: hypoxia leads to excitotoxic levels of
     glutamate, which cause a rise in intracellular calcium, which in turn,
     leads to neuronal death due to apoptosis or necrosis.  Neuroprotection is
     a process that attempts to preserve the cells that were spared during the
     initial insult, but are still vulnerable to damage.  Although not yet
     available, a neuroprotective agent would be of great use in arresting the
     progression of glaucoma.  There is evidence that neuroprotection can be
     achieved both pharmacologically and immunologically.  Pharmacological
     intervention aims at neutralising some of the effects of the nerve-derived
     toxic factors, thereby increasing the ability of the spared neurons to
     cope with stressful conditions.  On the other hand, immunological
     interventions boost the body's own repair mechanisms for counteracting the
     toxic effects of various chemicals generated during the cascade.  This
     review, based on a literature search using MEDLINE, focuses on diverse
     cellular events associated with glaucomatous neurodegeneration,
     and discusses some pharmacological agents believed to have a
     neuroprotective role in glaucoma.
SO   Journal of Postgraduate Medicine, (2003) Vol. 49, No. 1, pp. 90-95. .
     Refs: 47
     ISSN: 0022-3859  CODEN: JPMDA3
PY   2003

=>
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